#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									The study does not show a statistically significant increase in the risk or incidence of breast cancer (Dep. V., p.969, ll.1-2, 4-7). The study was flawed in the following ways: (1) it relied on death certificates, which have been shown to be inaccurate; (2) it did not have any real surrogate of dose because it lumped the people studied together; (3) it assumed that there was exposures to PCBs in the area without any attempt to figure out the dose; and (4) it included people who had less then a year of exposure (Dep. V, p.970, ll.5-25, p.971, p.1-20).
43	Cancer Mortality Among Workers in Chemical Plant Contaminated with Dioxin. Manz, A, et al., 1991.	Yes	Yes	**No**	**No**	**No**	**No**	Yes	Analyzed whether TCDD, a contaminant in the production of herbicides, is a human carcinogen by conducting a retrospective cancer mortality study. Only 7% of women worked in high exposure area of plant. Dr. Dahlgren acknowledged that: The study is concerned with TCDD and does not contain statistically significant data (Dep. IV, p.823, l.17). The study does not identify a dose level of 2, 3, 7, 8 – TCDD which is significant for increasing the risk of breast cancer (Dep. IV, p.824, l.6). The study does not document any exposure level (Dep. IV, p.824, ll.9-12).
44	Breast Cancer Incidence and Exposure to Environmental Chemicals in 82 Counties in Mississippi. Mitra, Amal, and	Yes	Yes	**No**	Yes	Yes	**No**	Yes	Analyzed patterns of breast cancer incidence in relation to environmental chemicals in Mississippi to assess environmental risks for breast cancer. Dr. Dahlgren acknowledged that: The study looked at the U.S. EPA's air and toxic release inventory data for Mississippi and did not identify Grenada County as a county with a higher risk

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	Faruque, Fazlay, 2004								or incidence of breast cancer (Dep. V, p.958, ll.21-24). The study evaluated overall pollution and not one pollutant or a series of pollutants (Dep. V, p.960, l.4).
45	Breast Cancer and Environmental Risks: Where is the Link? Mitra, Amal, et al. 2004	**No**	Yes	Yes	Yes	Yes	**No**	Yes	Analyzed relationship between wide assortment of environmental factors and breast cancer. Acknowledged that evidence regarding organochlorine exposure and breast cancer is mixed. Dr. Dahlgren criticized article as not doing "a particularly very good job" in summarizing all of the evidence or studies on organochlorines and PCBs (Dep. V, p.956, ll.14-21, 23-25, p.957, ll.1-2). Dr. Dahlgren acknowledged that: The article did not look at creosote and pentachlorophenol in isolation (Dep. V, p.957, l.8).
46	Epidemiological Factors of Cancer in California. Moran, Edgar, 1992	**No**	Yes	Yes	Yes	Yes	**No**	Yes	Analyzed ethnic, occupational, and lifestyle factors associated with risks of cancers in California. Dr. Dahlgren acknowledged that the article only dealt with risk factors for cancer in general (Dep. V, p.962, ll.18-23).
47	The Role of Aromatic Hydrocarbons in the Genesis of Breast Cancer. Morris, J.J., et al., 1992.	**No**	**No**	**No**	**No**	**No**	**No**	**No**	Analyzed the associations between aromatic hydrocarbons and cancers. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. IV, p.828, ll.4-14). The article does not address creosote as a mixture (Dep. IV, p.830, l.3). The article does not document any exposure levels to any particular PAHs (Dep. IV, p.830, l.6).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									The article does not calculate relative risk levels and does not identify any particular exposure level that is necessary to produce harm (Dep. IV, p.830, ll.8-11, 15).
48	Immunoperoxidase Detection of Polycyclic Aromatic Hydrocarbon-DNA Adducts in Breast Tissue Section. Motykiewicz, Grazyna, et al., 2001.	Yes	Yes	Yes	**No**	**No**	**No**	**No**	Analyzed the role of PAH in the etiology of breast cancer by monitoring the existence of PAH-DNA adducts in breast tissues collected from cancer patients and benign disease patients. Acknowledged that the small sample size was a major limitation and, therefore, its data should be interpreted with caution. None of the workers examined had occupational exposure to PAHs. Dr. Dahlgren criticized the study for making the mistake of analyzing cancer tissue instead of normal breast tissue (Dep. V, p.965, ll.2-4). Dr. Dahlgren stated, "I don't think it was a well-designed study" (Dep. V, p.966, ll.19-21). Dr. Dahlgren acknowledged that the study does not have much relevance for his opinions regarding Sherrie Barnes (Dep. V, p.966, ll.13-21).
49	Health Impact of Polychlorinated Dibenzo-p-dioxins: A Critical Review. Mukerjee, Debdas, 1997.	**No**	Yes	**No**	**No**	**No**	**No**	**No**	Analyzed animal and human data to identify any possible health effects associated with environmental exposures to PCDDs. Acknowledged that very little data was available to demonstrate causes and effects from exposure to dioxins. Dr. Dahlgren acknowledged that: He did not rely on this article to formulate his opinion regarding Sherrie Barnes (Dep. V, p.973, ll.7-13).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
50	Adipose Concentrations of Organochlorine Compounds and Breast Cancer Recurrence in long Island, New York. Muscat, Joshua, et al., 2003.	Yes	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed whether the estrogenic effects of *organochlorine compounds* ("OCs") *might* adversely affect breast cancer recurrence. Acknowledged that the levels of OCs (such as pesticides and PCBs) in breast tissue were not correlated with stage of breast cancer. Highest concentration of DDE was not a significant predicator of recurrence. Intermediate levels of some pesticides were associated with an increased risk relative to lowest levels, BUT associations were not dose dependent. Stated, "[t]here were relatively few events in this study, and the positive findings could have been due to bias. . . . There are an insufficient number of studies to determine whether our results are consistent with the literature." (p.1477) Dr. Dahlgren acknowledged that: There was a previous "negative" paper published by the same authors of this article (Dep. IV, p.832, l.21). Other studies he referenced contrast with this study by finding that the same congeners did not increase the risk of breast cancer (Dep. IV, p.833, ll.14-15). The study does not identify an exposure level necessary to cause harm (Dep. IV, p.834, l.18). The study recognized that there were relatively few events in the study and that the positive findings could have been due to bias (Dep. IV, p.834, l.22).
51	Environmental Exposure to Polychlorinated Biphenyls (PCBs) and Breast Cancer: a Systematic Review of the Epidemiological	**No**	Yes	**No**	Yes	**No**	**No**	**No**	Analyzed epidemiological studies on the relation between environmental exposure to PCB's and risk of breast cancer. Vast number of studies reviewed did not find any association between total PCB concentrations and breast cancer risk.

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	Evidence. Negri, E., et al., 2003.								Dr. Dahlgren acknowledged that: The article is a review paper (Dep. IV, p.836, l.8). The article concluded, "the epidemiological evidence does not support the hypothesis of a direct relation between environmental exposure to PCB adulthood in the general population and the risk of breast cancer." (Dep. IV, p.837, ll.3-4). The article is generally informative but not directly related to Sherrie Barnes (Dep. IV, p.838, l.4).
52	Age Specific Dioxin TEQ Reference Page. Patterson, Donald, et al., 2004.	Yes	Yes	**No**	**No**	**No**	**No**	**No**	Analyzed pre-existing data on serum dioxin levels in four studies to calculate age specific reference range levels to determine the relationship between overall blood dioxin TEQs and TCDD with respect to age. Dr. Dahlgren acknowledged that the only relevant part of the study was its point that TEQs rise with age (Dep. V, p.976, ll.7-8).
53	Mixtures of Four Organochlorines Enhance Human Breast Cancer Cell Proliferation. Payne, Joachim, et al , 2001.	Yes	Yes	**No**	**No**	Yes	**No**	**No**	Analyzed the effect of environmental estrogenic chemicals on in vitro breast cancer cells. Dr. Dahlgren acknowledged that none of the organochlorines analyzed in the study are commonly seen in pentachlorophenol (Dep. V, p.1045, ll.20-22).
54	Carcinogen-DNA Adducts in Human Breast Tissue. Perera, Frederica, et al., 1995.	Yes	Yes	Yes	**No**	Yes	**No**	**No**	Analyzed whether adducts of the type formed by pervasive environmental carcinogens could be detected in breast tissue. Acknowledged that a limitation of the study is that smoking status was not available for the controls. Acknowledged that although the difference between smokers and non-smokers in terms of DRZ was statistically significant the small number of subjects and the fact that the non-breast cancer patients were not comparable in terms of age and other characteristics to

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									the cases precluded any inference as to causality. Acknowledged that PAHs are found in cigarette smoke, ambient air, drinking water, food along with workplace environments. Dr. Dahlgren acknowledged that the study was not trying to prove or disprove causality (Dep. V, p.992, ll.5-8).
55	Molecular Epidemiology in Environmental Carcinogenesis. Perera, Frederica, et al., 1996.	**No**	Yes	**No**	**No**	**No**	**No**	Yes	Analyzed epidemiologic research on the use of biologic markers in the prevention of environmental carcinogenesis and reproductive toxicity. Does not provide detailed information about breast cancer. Does not analyze exposure to creosote or pentachlorophenol. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.1061, l.3). The article is not relevant to Sherrie Barnes. (Dep. V, p.1066, ll.13-22).
56	Molecular Epidemiology: On the Path to Prevention Perera, Frederica, et al., 2000.	**No**	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed various environmental exposures and their roles in lung, breast, and liver cancers and leukemia. Dr. Dahlgren acknowledged that the article was more of a policy paper discussing the prevention of certain diseases in the future and not an analysis of what is currently causing those diseases. (Dep. V, p.979, ll.11-14, ll.18-23).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
57	Occupational Risk Factors for Breast Cancer Among Women in Shanghai. Petralia, Sandra, et al.,1998	Yes	Yes	**No**	Yes	Yes	**No**	Yes	Analyzed occupational risk factors associated with breast cancer in Chinese women, including exposure to certain chemicals. Dr. Dahlgren acknowledged that: The study was relevant to the present case because it studied the health effects of benzene exposure on a group of women. In addition, he acknowledged that Sherrie Barnes exposure to benzene from the Koppers plant had not been measured, modeled, or calculated (Dep. VI, p.1084, ll.7-21, p.1085, ll.2-12). The study does not indicate what level of exposure is necessary to increase the risk of breast cancer (Dep. VI, p.1085, 11.16-23).
58	Risk of Pre-menopausal Breast Cancer in Association with Occupational Exposure to Polycyclic Aromatic Hydrocarbons and Benzene. Petralia, Sandra, et al., 1999.	Yes	Yes	**No**	Yes	**No**	**No**	**No**	Analyzed the relationship between risk of pre-menopausal breast cancer and occupational exposure to benzene and PAH and whether proposed relationship between PAH and breast cancer differed by tumor estrogen receptor status. Contained risk ratios for pre-menopausal breast cancer associated with having occupational exposure to PAH and benzene. Dr. Dahlgren acknowledged that the study does not identify an exposure level that is necessary to cause harm (Dep. IV, p.843, 1.7).
59	Deregulation of Cell Proliferation by Polycyclic Aromatic Hydrocarbons in Human Breast Carcinoma MCF-7 Cells Reflects Both Genotoxic and Nongenotoxic Events. Pliskova, Matina, et	Yes	Yes	**No**	**No**	**No**	**No**	**No**	Analyzed whether activation of ER by PAHs or their metabolites could induce cell proliferation in estrogen-sensitive cells. Acknowledged that questions remained about whether the proliferation effects of the two particular PAHs studied were restricted to the particular cell line studied or whether it was of any in vivo relevance. (p.255) Dr. Dahlgren acknowledged that: The study does not identify a particular dose or

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	al., 2005.								exposure level in which harm would occur (Dep. IV, p.847, ll.13-14). The study looked at two particular PAHs, and not a creosote mixture (Dep. IV, p.847, ll.23, 25, Expert Report, p.42). However, Dr. Dahlgren claimed in his expert report that this study indicated a causal link between breast cancer and the chemicals of interest in this case. (Expert Report, p.42, p.116). The study does not actually analyze the synergistic effect between PAHs and TCDD (Dep. IV, p.848, l.11).
60	Dioxin Exposure and Public Health in Chapaevsk, Russia. Revich, B, et al., 2001.	Yes	Yes	**No**	**No**	Yes	**No**	Yes	Analyzed public health of individuals exposed to dioxins in the water, air, and cow's milk in Chapaevsk, Russia. Analyzed chemicals not at issue in the present case. Dr. Dahlgren acknowledged that the study is not a cohort study or a control study (Dep. IV, p.851, ll.22-25).
61	The Association between Glutathione S-Transferase M1 Genotype and Polycyclic Aromatic Hydrocarbon-DNA adducts in Breast Tissue. Rundle, Andrew, 2000.	Yes	Yes	Yes	**No**	**No**	**No**	**No**	Analyzed DNA damage resulting from exposure to PAHs and susceptibility attributable to inherited deletion of the xenophobic detoxifying gene.

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
62	The Relationship Between Genetic Damage from Polycyclic Aromatic Hydrocarbons in Breast Tissue and Breast Cancer. Rundle, Andrew, et al., 2000.	Yes	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed whether exposure and susceptibility to PAH, as measured by PAH-DNA adducts in breast tissue, are associated with human breast cancer. Stated, "A major limitation was [its] inability to determine the specific contributions of all of the possible PAH exposure sources, both past and present, to the PAH-DNA adducts measured in breast tissue." In addition, it stated "[a]nother concern is that the use of benign breast disease controls may have biased the study results towards the null." (p.1287) Dr. Dahlgren acknowledged that the study does not definitively demonstrate that PAH-DNA adducts play a role in the further progression of malignant cells. (Expert Report, p.42). Dr. Dahlgren acknowledged that: It was a mistake or weakness for the study to compare patients with benign breast tissue with cancer patients (Dep. V, p.997, 14-20).
63	Molecular Epidemiologic Studies of Polycyclic Aromatic Hydrocarbon-DNA Adducts and Breast Cancer. Rundle, Andrew, et al., 2002	**No**	Yes	Yes	Yes	Yes *See Comments*	**No**	**No**	Analyzed the role of PAH-DNA adducts in breast cancer. It also analyzed the reliability of the scoring procedures used with immunohistochemical assay for PAH-DNA adducts and of potential bias arising from the use of benign breast disease (BBD) controls. Acknowledged that there was potential bias due to the use of BBD controls especially because BBD controls may overestimate the prevalence of family history of breast cancer compared to healthy controls. Also acknowledged that the analyses showed that the technician had a significant effect on scoring in regards to tumor and non-tumor tissue. Dr. Dahlgren acknowledged that the article is a review paper (Dep. V., p.1000, ll.24-25). *Statistical Significance: Due to modest sample size the finding that increased levels of PAH-DNA adducts in*

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									breast tissue were associated with breast cancer case-control status is statistically significant only when adducts levels in tumor tissue are compared to those seen in benign tissue
64	The Interaction Between Alcohol Consumption and GSTM1 Genotype on Polycyclic Aromatic Hydrocarbon-DNA Adduct Levels in Breast Tissue. Rundle, Andrew, et al., 2003.	Yes	Yes	Yes	**No**	**No**	**No**	**No**	Analyzed the association between alcohol consumption, GSTNI genotype, and PAH-DNA adduct levels in breast tissue. Does not focus on whether PAH's cause breast cancer. It looks at associations between breast cancer, alcohol, and the formation of PAH-DNA adducts. Nevertheless, Dr. Dahlgren claimed in his expert report that this study indicated a causal link between breast cancer and the chemicals of interest in this case. (Expert Report, p.116). Stated, "a concern is that the BBD [benign breast disease] controls may overly share risk factors, both measured and unmeasured, with the cases. . . the use of BBD controls will cause risk estimates for the exposure to be attenuated to the null." (p.913) Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the particular polymorphism analyzed in the study (Dep. V, p.1069 l.20).
65	Interaction between Genetic Polymorphism of Cytochrome P450-1B1 and Environmental Pollutants in Breast Cancer Risk. Saintot, M., et al., 2004	Yes	Yes	Yes	Yes	Yes	**No**	**No**	Analyzed the interaction between a particular polymorphism and environmental exposure by examining Caucasian women who lived near to a waste incinerator. Dr. Dahlgren acknowledged that he is unaware whether Sherrie Barnes had the particular polymorphism that is discussed in the study (Dep. V, p.1006, l.10).
66	Reduced DNA Repair of Benzo[*a*]pyrene Diol	Yes	Yes	Yes	**No**	**No**	**No**	**No**	Analyzed whether reduced DNA repair is associated with risk of breast cancer in women and whether the risk may be modulated by polymorphisms of DNA

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	Epoxide-Induced Adducts and Common XPD Polymorphisms in Breast Cancer Patients. Shi, Qiuling, et al., 2004.								repair genes. Study was small and preliminary. Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the particular DRC level identified in the study (Dep. V, p.1011, ll.14-15).
67	Genetic Susceptibility to Cancer. Spitz, Margaret & Bondy, Melissa, 1993	**No**	Yes	Yes	**No**	Yes	**No**	**No**	Analyzed studies examining mutagen sensitivity in cancer patients and relatives. Dr. Dahlgren acknowledged that article is generally informative but not related to causation (Dep. V, p.1033, l.4).
68	Cancer, Heart Disease, and Diabetes in Workers Exposed to 2,3,7,8 Tetrachlorodibenzo-p-dioxin. Steenland, Kyle, et al., 1999.	Yes	Yes	**No**	**No**	Yes	**No**	Yes	Analyzed the link between cancer and TCDD exposure in four industrial cohorts. Study does not address breast cancer. Nevertheless, Dr. Dahlgren cites this study in his expert report to suggest that TCDD may initiate and promote breast cancer. (Expert Report, p.116). Dr. Dahlgren acknowledged that the study is not relevant to the question of breast cancer (Dep. V, p.1053, ll.3-5).
69	Breast Cancer Risk in Relation to Adipose Concentrations of Organochlorine Pesticides and Polychlorinated Biphenyls in Long Island, New York. Stellman, Steven, et al., 2000.	Yes	Yes	Yes	Yes	**No** *See Comments*	**No**	**No**	Analyzed the possible etiological role of organochlorine compounds in breast cancer development on Long Island. Stated, "[t]he present analysis for the Long Island population is consistent with numerous studies in other populations that have shown little association between OCC body burden and breast cancer risk." (p.1245) Dr. Dahlgren acknowledged that: The study did not find an association between breast cancer and PCB congener 118 or 153 (the most abundant di-ortho congener). The study only found an association between breast cancer and PCB congener

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									183 (Dep. V, p.87, ll.4, 8, 12-16). The study is not directly related to his conclusions regarding Sherrie Barnes (Dep. V, p.877, ll.7-12). The study is a negative study and, therefore, does not support his conclusions regarding Sherrie Barnes (Dep. V, p.877, ll.18-20). *Statistical Significance: Article stated, "[t]he large number of statistical tests that can be carried out in this database may produce a few statistically significant findings that have little or no biological meaning."*
70	Health Effects of Chlorophenol Wood Preservatives on Sawmill Workers. Sterling, Theodore, et al., 1982.	Yes	Yes	Yes	**No**	**No**	**No**	Yes	Analyzed and surveyed wood workers for health complaints possibly associated with chlorophenol exposure. Dr. Dahlgren acknowledged that the study does not relate to cancer (Dep. V., p.977, l.16).
71	Formation and Persistence of Benzo(a)pyrene Metabolite-DNA Adducts. Stowers, S., & Anderson, M., 1985	**No**	**No**	**No**	**No**	Yes	**No**	**No**	Analyzed the effect of BP and PAHs on various rat and rabbit metabolic activities. Dr. Dahlgren acknowledged that: The article is a review paper on animal studies (Dep. V, p.1033, ll.17-25, p.1034, ll.1-6). The article is generally informative of the causation issues in animals (Dep. V, p.1035, l.7).
72	Polymorphisms in the DNA Repair Enzyme XPD are Associated with Increased Levels of PAH-DNA Adducts in a Case-Control Study of Breast Cancer. Tang, Deliang, et al.,	Yes	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed the associations between DNA adduct levels in breast tissue, risk of breast cancer, and polymorphisms in the DNA repair enzyme XPD. Indicated that it is the first report on XPD and breast cancer and its data does not suggest that XPD polymorphisms are associated with breast cancer. Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the XPD polymorphism

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	2002								analyzed in the study (Dep. V, p.1014, l.18).
73	Sulftransferase 1A1 (SULT1A1) Polymorphism, PAH-DNA adduct levels in Breast Tissue and Breast Cancer Risk in a Case-Control Study. Tang, Deliang, et al, 2003.	Yes	Yes	Yes	**No**	**No**	**No**	**No**	Analyzed associations between genetic susceptibility due to inherited polymorphisms of the Phase II detoxification enzyme SULT1A1, breast cancer risk, and PAH-DNA adducts. Acknowledged that its small sample size limited its statistical power to detect modest odds ratios. Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the particular polymorphism analyzed in the study (Dep. V, p.1016, l.20).
74	Cigarette Smoking and the Risk of Breast Cancer in Women: A Review of the Literature. Terry, Paul, et al., 2002.	**No**	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed links between breast cancer and cigarette smoking. Indicated that the studies that suggest that there is a link between breast cancer and cigarette smoking require confirmation in future studies. Dr. Dahlgren acknowledged that the article is not directly relevant to his opinion regarding Sherrie Barnes (Dep. V, p.993, ll.1-10).
75	Bulky DNA Adducts and Risk of Cancer: A Meta-Analysis. Veglia, Fabrizio, et al., 2003	Yes	Yes	Yes	**No**	Yes	**No**	**No**	Analyzed case-control studies that linked current smoking to increased lung and bladder cancer. Dr. Dahlgren acknowledged that the study does not address breast cancer (Dep. V, p.1035, l.25).
76	A Novel Effect of Dioxin: Exposure during Pregnancy Severely Impairs Mammary Gland Differentiation. Vorderstrasse, Beth, et al., 2004.	Yes	**No**	**No**	**No**	Yes	**No**	**No**	Analyzed whether the extensive differentiation of mammary tissue that occurs during pregnancy is also sensitive to disruption by AhR activation. Does not relate to breast cancer. Dr. Dahlgren acknowledged that: The study does not directly study mammary gland

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									cancer or breast cancer (Dep. IV, p.697, ll.6-9). The study does not give any relative risk data for breast cancer (Dep. IV, p.698, l.4). The study analyzed TCDD in isolation (Dep. IV, p.698, l.6).
77	Lipid Peroxidation-induced putative Malondialdehyde-DNA Adducts in Human Breast Tissues. Wang, Mianying, et al., 1996.	Yes	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed whether oxidative stress and lipid peroxidation have been suggested to play a role in breast carcinogenesis. It is a pilot study. Acknowledged that diet, smoking, alcohol consumption, certain disease status, along with carcinogen exposure (and other factors) has been associated with the extent of lipid peroxidation in animal models and in other human cancers. In addition, it acknowledged that it did not have detailed information on many of these factors when conducting the study. Acknowledged that the observed lower levels of adducts could be due to tissue difference as well as technical difference. As a result, the article stated, "It has been previously demonstrated that the MDA-dG adduct is susceptible to nuclease P1 digestion. Therefore, some adducts could have been lost from detection during the analysis, and the observed levels of these adducts . . . might represent a minimum estimate of the actual levels."(p.709) Dr. Dahlgren acknowledged that: The study is not as important as the other papers he cites (Dep. VI, p.1078, ll.20-21). The study is only relevant to show "an interesting observation that there [are] some other mechanisms by which cancer can be induced; mainly, this so-called lipid peroxidation" and its data on the relationship

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
									between benzo(a)pyrene. In addition, he acknowledged that he does not know whether Sherrie Barnes had lipid peroxidation-induced DNA adducts (Dep. VI, p.1078, ll.20-25, p.1080, ll.1-2, 6).
78	Serum Dioxin Concentrations and Breast Cancer Risk in the Seveso Women's Health Study. Warner, Marcella, et al., 2002	Yes	Yes	**No**	Yes	**No**	**No**	Yes	Analyzed the association between individual serum TCDD levels and breast cancer risk in women residing around Seveso, Italy, in 1976, at the time of a big industrial explosion that exposed the population to high amounts of TCDD. Does not prove that TCDD was the cause of breast cancer. Contained small number of breast cancer cases in study. Stated, "[its] result should be considered an early finding because the number of cases [were] small and the cohort [was] relatively young."(p.628) Dr. Dahlgren acknowledged that: The range of serum TCDD levels in the women in the Seveso study was much higher then background exposure (Dep. V, p.888, l.6).
79	Comments on the History and Importance of Aromatic Heterocyclic Amines in Public Health. Weisburger, John, et al., 2002.	**No**	Yes	**No**	**No**	**No**	**No**	**No**	Analyzed the history and importance of aromatic heterocyclic amines in public health. Stated, "[e]pidemiological data suggests that meat eaters may have a higher risk of breast and colon cancer."(Abstract) Dr. Dahlgren stated, "I think this paper probably doesn't have to be included in our list. It is a little bit further from the main topic of our case here." (Dep. VI, p.1080, ll.12-14).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
80	Associations of Cancer Site and Type with Occupation and Industry from the Third National Cancer Survey Interview. Williams, Roger, et al., 1977	Yes	Yes	Yes	Yes	Yes	**No**	**No**	Analyzed cancer incidence by occupation after controlling for assorted risk factors such as smoking. Found breast cancer more common for women in certain occupations.
81	Pesticides-How Research Has Succeeded and Failed in Informing Policy: DDT and the Link with Breast Cancer. Wolff, Mary, 1995.	**No**	Yes	**No**	**No**	**No**	**No**	**No**	Analyzed possible link between exposures to environmental chemicals and breast cancer. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.1042, l.9-17). He agrees with the article's statement that, "[e]xisting methodologies are often inadequate to study complex diseases like cancer, reproductive dysfunction and neurotoxicity, especially when attempting to link subtle biological effects with complex and low-level exposures" (Dep. V, p.1044, ll.9-18).
82	Breast Cancer and Environmental Risk Factors: Epidemiological and Experimental Findings. Wolff, Mary, et al., 1996	**No**	Yes	Yes	Yes	Yes	**No**	Yes	Analyzed a wide variety of risk factors associated with breast cancer. Dr. Dahlgren acknowledged that the article is a review paper (Dep. VI, p.1087, ll.17-25, p.1088, ll.1-3).
83	Sensitivity to Benzo(a)pyrene Diol-Epoxide Associated with Risk of Breast Cancer in Young Women and Modulation by Glutathione *S*-Transferase	Yes	Yes	Yes	Yes	**No**	**No**	**No**	Analyzed whether breast cancer patients may be sensitive to tobacco-induced carcinogenesis and whether this sensitivity could be modulated by variants of metabolic genes. Acknowledged that the study suffered from the following limitations: (1) hospital based and ,therefore, may not have provided generalizable findings to the general population; (2) relatively small sample and

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
	Polymorphisms: A Case-Control Study. Xiong, Ping, et al., 2001.								used multiple comparisons; and (3) detailed information on alcohol consumption from its study population was not available. Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the particular polymorphism analyzed in the study (Dep. V, p.1020, l.21).
84	Serum Polychlorinated Biphenyls, Cytochrome P-450 1A1 Polymorphisms, and Risk of Breast Cancer in Connecticut Women. Zhang, Yawei, et al., 2004.	Yes	Yes	Yes	Yes	**No** *See Comments*	**No**	**No**	Analyzed the potential effect of gene-environment interaction between CYP1A1 and serum PCB levels on breast cancer risk among Caucasian women in Connecticut. The article acknowledged that the study suffered from the following limitations: (1) small number of subjects; (2) included patients with benign breast disease in the control group; (3) used serum levels of PCBs rather than adipose tissue levels and that may not reflect the real body burden of PCBs; and (4) current levels of PCBs may not have represented the levels the subjects had when their disease developed. *Statistical Significance: Article stated, "a significantly increased risk of breast cancer was found only for the CYP1A1 m2 genotype."* Dr. Dahlgren acknowledged that: The study mistakenly used women with benign breast disease as controls instead of women who had not been diagnosed with any type of breast disease, which confounded the study "rather significantly" (Dep. V, p.893, l.25, p.894, ll.1-3, 8-9). The study can be described as an incomplete step in the direction of evaluating the connection between organochlorine exposure and various polymorphisms (Dep. V, p.894, ll.23-24).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
85	Detection of 2-Amino-1-Methyl-6-Phenylimidazo[4,5-b]-Pyridine-DNA Adducts in Normal Breast Tissues and Risk of Breast Cancer. Zhu, Jijiang, et al., 2003.	Yes	Yes	Yes	Yes	Yes	**No**	**No**	Analyzed PhIP-DNA adducts in breast tissue from women having unknown exposure to HCAs. In his deposition, Dr. Dahlgren did not state that the study is relevant regarding causation in the Sherrie Barnes case but instead acknowledged that the article simply stands for the theoretical proposition that PAHs form into nitrosamine-type compounds or heterocyclic amines (Dep. VI, p.1082, ll.6-18). Dr. Dahlgren acknowledged that this study dealt with the effect of certain compounds, found in cooked meats, and their potential for increasing breast cancer risk (Dep. VI, p.1082, ll.24-25, p.1083, ll.1-17).
86	Environmental "Endocrine Disrupters" Get a Global Look. Ziegler, Jan, 1997	**No**	**No**	**No**	**No**	**No**	**No**	**No**	Editorial/news story Dr. Dahlgren stated, "I don't remember why I included this [article] . . . It doesn't have any information in it that is particularly useful." (Dep. V, p.1023, ll.2-4).

*** "Yes"- includes human studies, in vitro studies, or review articles that analyzed human studies.**

**** "Yes"- includes articles or studies that contained statistically significant data for any cancers.**